

was seen. Biopsy specimens showed a polymorphic infiltration of the dermis without necrosis. Stains for bacterial or fungal infection and cultures for virus and mycobacteria were negative. Thalidomide was administered (100 mg twice a day) and the patient dramatically improved within 48 hours of treatment. Thalidomide was interrupted 6 days after the beginning of treatment because of transient paraesthesias and pain involving the four limbs. Clinical examination disclosed sensory and motor peripheral neuropathy, confirmed by electromyogram with decreasing motor conduction in the four limbs. The CSF yielded a lymphocytic aseptic meningitis. Neurologic abnormalities spontaneously regressed one month later.

Thalidomide is of value in the treatment of many skin conditions including genital ulcerations and should be tried for resistant aphthous ulcers in HIV-infected patients.² Most of the side effects of thalidomide therapy are minor including fatigue, constipation, nausea and dry skin. Hypersensitivity reactions (fever, erythematous macular rash) have been recently reported in three HIV-infected patients who received thalidomide for aphthous oropharyngeal ulcerations.³ The side effects of thalidomide seem to be more severe and less tolerated in HIV-infected patients.

Another major complication is neuropathy which is dose dependent,⁴ of an axonal type affecting mainly sensory fibres of the lower limbs. Usually motor conduction changes are not found. In our patient neuropathy cannot be attributed to thalidomide because of the short period before occurrence, motor conduction changes and CSF abnormalities. His neuropathy may have been due to the HIV-infection itself.

As the benefits greatly outweigh the risks of side effects of thalidomide treatment, especially compared with other possible regimens, this medication should be considered for the treatment of genital and aphthous ulcers before immunosuppressive agents like corticosteroids in the treatment of HIV-infected patients.

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HIV infection and menstrual abnormalities

The number of women infected by human immunodeficiency virus (HIV) is increasing.¹ The incidence of lower genital tract intra-epithelial neoplasia and infection has been well documented.^{2,3} There are, however, few data on menstrual abnormalities in these women. Reports of an increased prevalence of amenorrhoea⁴ need further evaluation since factors such as substance misuse, prescribed drugs such as methadone and weight loss can significantly affect the menstrual cycle. Possible HIV-related causes include premature ovarian failure, secondary to opportunistic infections such as cytomegalovirus⁵ or autoimmunity resulting from the polyclonal gammopathy associated with HIV infection.⁶

To assess the prevalence of menstrual dysfunction in HIV seropositive women a retrospective analysis was performed of the menstrual histories from women attending two genitourinary clinics in London. Those patients who were pregnant, post menopausal or on hormonal contraception were excluded from analysis. Of the 58 women remaining, 42 (72.4%) had regular menstrual cycles, 10 (17.2%) had oligomenorrhoea and six (10.4%) had amenorrhoea. Analysis of these data revealed that of the women with amenorrhoea, two were current intravenous drug users and four were on methadone maintenance. Similarly, of the women with oligomenorrhoea, two were current intravenous drug users, four were on methadone maintenance and three were taking cocaine.

Our findings initially suggested a higher prevalence of amenorrhoea and oligomenorrhoea in HIV seropositive women in comparison with the general population.⁷ However, this difference is not sustained once substance misuser's and patients on methadone maintenance have been excluded. The number of women with severe immunosuppression (CD4 < 200) in this study was less than 10% and symptomatology may change as increasing numbers of women survive prolonged periods of immunosuppression. A continuing long term prospective study of this subject, with case matched controls is currently being performed.

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